LITHIUM CLEARANCE IN COMPENSATED AND DECOMPENSATED CARDIAC PATIENTS

رسالة

Thesis

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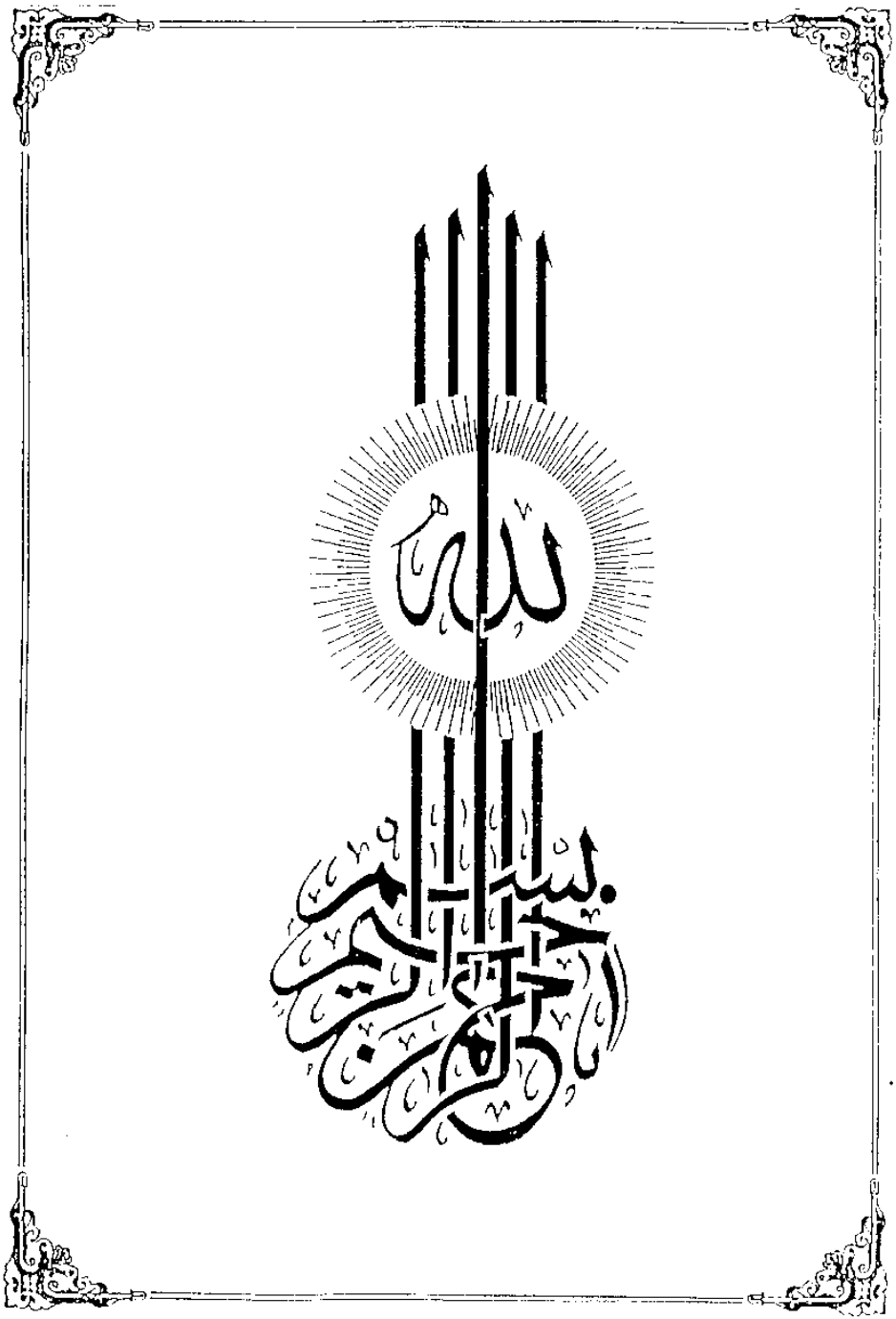
بسم الله الرحمن الرحيم

﴿قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ﴾

«صدق الله العظيم»
(سورة البقرة آيه رقم (٣٢)



TO...

MY PARENTS

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Abbreviations

A.N.P	Atrial natriuretic peptide
AR	Aortic regurge or AI= Aortic incompetence
AS	Aortic stenosis
ASD	Atrial septal defect
ASO	Anti-streptolysin O
CHD	Congenital heart disease
C.H.F.	Congestive heart failure
G.F.R.	Glomerular filtration rate
MR	Mitral regurge or MI= Mitral incompetence
MS	Mitral stenosis
T.R.	Tricuspid regurge
PDA	Patent ductus arteriosus
PS	Pulmonary stenosis
RF	Rheumatic fever
RHD	Rheumatic heart disease
VSD	Verticular septal defect
S.L.E.	Systemic lupus erythematosus
P.A.N.	Poly arteritis nodosa
H.B.	Hemoglobin
B.U.N.	Blood urea nitrogen

INTRODUCTION AND AIM OF THE WORK

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Lithium is a monovalent cation which belongs to the group of alkaline metals together with sodium and potassium with which it shares some of their properties. It is excreted mainly in urine, 80% of the filtered lithium is reabsorbed probably by the same mechanism responsible for sodium reabsorption in the proximal renal tubules *(Bladessarini and Lipinski, 1975).*

Since the proximal reabsorption of these two ions is competitive, So any change in sodium level or renal tubular handling of it is suggested to affect lithium clearance *(El-Feky et al., 1992).*

In diseases characterized by sodium retention as heart failure, there is abnormal handling of Na by the kidney with tendency to retain it with water. This tendency for Na retention is hazardous to the patients and must be considered in their management.

In this work, it is aimed to study the renal sodium

handling in cardiac patients some are compenated and some with heart failure using lithium clearance which is a quantitative method to estimate proximal sodium delivery *(Strazzullo et al., 1988).*

REVIEW OF LITERATURE

HEART FAILURE

Definition of heart failure:

Heart failure in infancy and childhood represents a clinical syndrome which reflects the inability of the myocardium to meet the metabolic requirements of the body including those needs of the growth process *(Talner, 1983).*

Friedman and George (1984), defined heart failure as the inability of the heart to pump blood in a quantity commensurate with the body requirements. This may be due to impaired myocardial performance, excessive hemodynamic burden or a combination of both. Sometimes, heart failure is associated with an inability of the heart to increase its output adequately to meet increased demands of tissues with effort *(Keith, 1978).*

Frequently this syndrome is encountered in the pediatric age group especially in infancy *(Talner, 1983).*

Heart failure is not a syonymous with circulatory failure which means inadequacy of the cardiovascular system of providing oxygen and nutrition to the tissues and removing metabolic products from them. This may be caused by either cardiac or peripheral conditions e.g decreased venous return or

inadequate blood volume *(Jaiyesimi, 1981)*. Latent heart failure is said to be present when heart failure is absent at rest but is apparent only during period of increased stress as emotions, exercise and fever *(Schalant et al., 1982)*.

Eric et al, (1993) defined congestive heart failure as a progressive disorder of systolic and diastolic ventricular dysfunction that results in reduced forward flow and increased filling pressures.

Another definition of congestive heart failure that it is a clinical syndrome characterized by congestion of the pulmonary or systemic circulation owing to increased pulmonary or systemic venous pressure to the level of plasma protein oncotic pressure or above *(Rashed, 1987)*. This congestion must be cardiac in origin to distinguish it from non cardiac circulatory overload or congestion which may result from increased blood volume e.ge acute glomerulonephritis or increased venous return e.g A.v fistula, severe anemia. Many patients with non cardiac circulatory overload eventually develop secondary high output heart failure *(Schlant et al., 1982)*.

Incidence of heart failure in infancy and childhood:

At a pediatric Hospital in Toronto, 20% of 10535 children